I would emphasize his observation that the general nutritional picture of the child has much to do with efficiency in function of the feet. General strength and good vitality usually make certain such qualities in the feet. I am glad to see him emphasize the fact that shoes are worn as gloves for the normal feet. The most flexible shoe possible is ideal for the normal foot of the rapidly developing child.

Do not, however, some of Doctor Patton's ideas smack somewhat of nihilism? If heel-lifts, orthopedic shoes, and exercises do little or no good, then it seems that we are left with very little to do for these minor difficulties, except to correct the general nutrition of the child. I believe we would be amiss if these simple, and at least harmless, procedures were omitted in indicated cases. Perhaps the child would grow out of such minor difficulties as pronated and flat feet and knock-knees if we did nothing. However, if we failed to use these corrections, the blame would be ours should the child not make these adjustments. I have seen too many children rapidly and permanently improve when these simple, indicated corrections were made, to be willing to call them fallacies. I believe them to be too valuable as aids in the problem of nutrition and development to discard them.

*

Doctor Patton (Closing).—Doctor Sweet's discussion adds significant things which time would not allow in the original.

Doctor Harding—and San Diego—are to be congratulated if their community does not face, as does Los Angeles particularly, the widespread influence of confrères and lay bodies whose ideas are in accord with the fallacies which he so euphemistically terms "straw men."

Doctor Moody is not disagreeing, because there cannot be any possible objection to utilization of the simple measures he mentions; especially if—as I am sure is automatic in Doctor Moody's practice—the proper medical care is given at the same time.

PERIPHERAL ARTERIES: THEIR IMPORTANCE IN INDUSTRIAL PRACTICE*

By Felix Pearl, M.D.
San Francisco

Discussion *by Vernon P. Thompson, M. D., Los Angeles; Emile Holman, M. D., San Francisco; William J. Kerr, M. D., San Francisco.*

During the past few years the attention of the profession has been focussed, more than ever before, on the necessity for a clearer conception of the nature and course of affections of the peripheral arteries. The resultant investigations have borne fruit, so that at the present time the diagnosis and treatment of these affections rests upon a firmer and more accurate basis than was ever dreamed of previously.

There is no doubt that many insurance companies are being heavily taxed by the financial burden of patients who are suffering from manifest or occult chronic arterial disease, and that many of these patients are losing the benefit which early diagnosis and rational treatment offer them, not only for the present, but for the future as well.

In discussing the rôle of the peripheral arteries as related to industrial practice, one may divide the subject into two broad phases: (1) Direct injuries to previously normal peripheral arteries, and (2) industrial accidents occurring in individuals who have chronic arterial disease.

* From the Clinic of Sympathetic and Vascular Surgery, Mount Zion Hospital.
Read before the Industrial Medicine Section of the California Medical Association at the sixty-fifth annual session, Coronado, May 25-28, 1936.

INDICATIONS AND TREATMENT

Contusions and lacerations of large arteries are the most common direct injuries. If the main artery to an extremity is completely severed, the vessel should be immediately sutured if it is possible to approximate the ends. If it is cut two-thirds through, it should be converted into a complete section. The Carrell technique is used in our department of sympathetic and vascular surgery. Only three weeks ago, the writer successfully performed circular arteriorrhaphy of a severed brachial artery in a child of nine years. If suture is impracticable, it may be necessary to transplant a section of vein or to ligate the artery. For arterial ligation silk or tape is recommended, which is about one-fourth the diameter of the vessel to be ligated. According to the "Golden Rule" of Matas, the artery should be ligated directly at the bleeding point rather than in continuity at a distance from the injured site. I follow the practice of anchoring the main ligature by a mattress suture of fine silk, which passes through all the coats of both walls of the artery and embraces the ligature. This prevents the ligature from blowing off. Whenever the main artery to a normal extremity must be ligated, the question arises as to the desirability of ligating the main vein at a higher level. This should be determined in the individual case at the time of operation, utilizing the response to the Henle-Coenen and the Von Frisch tests for the competency of the collateral circulation, rather than by any set rule to ligate or not to ligate in every instance. To secure permanent ligation the vessel should always be transected between ligatures, and the cut end separated. When the artery is cut, one obtains the same effect as by periarterial sympathectomy in temporarily eliminating vascular spasm, which is often a factor of tremendous importance in the clinical picture of ischemia. The use of suction pressure therapy in cases which show evidence of dangerous vascular insufficiency may also be considered. Acute traumatic thrombosis of the main artery is a difficult problem. Here, opening the artery and attempting to remove the thrombus are at times indicated if tissue death is apt to ensue. At operation periarterial sympathectomy should be performed above the site of thrombosis to eliminate vascular spasm for a period of several weeks, during which time a competent circulation may have developed. At the same time, if it is considered wise, the main vein may be divided proximal to the site of arterial injury.

Large arteries may be eroded by processes secondary to trauma, which attack the wall from without, such as abscesses, cellulitis, and thrombophlebitis. Recently, a patient was referred who had had an extensive traumatic thrombophlebitis of the inner aspect of the right thigh, complicated by multiple pulmonary infarcts and followed by a sudden hemorrhage from an eroded femoral artery. The surgeon in charge acted promptly, and ligated in continuity the femoral artery above the site of erosion. At the present time, fifteen months after ligation, the dorsalis pedis pulsation is just felt, the extremity is warm and well nourished,

and vasomotor studies show a normal vasodilatation level. He has symptoms due to spasm of the arteries of this extremity brought on by exercise, which prevents him from working. These symptoms are not produced during the time that the homolateral lumbar sympathetic chain is blocked with novocain. I have advised lumbar ganglionectomy to relieve this disabling arteriospasm. Abnormal arteriovenous communications may occur from puncture wounds which injure simultaneously the vein and adjacent artery. These interfere with the competency of circulation of the extremity and throw a severe load on the heart. The collateral circulation which results from arteriovenous aneurysm is tremendous, and there is little danger in attacking these surgically if one waits from four to six months. The ideal treatment is ligation of all the vessels, arterial and venous, which open into the sac, and removal of the aneurysm under scrupulous asepsis. This differs from the treatment of traumative aneurysms in which the sac should be preserved to keep valuable collateral vessels intact.

We now come to the consideration of the patient with chronic arterial disease. The usual type of history and the methods of study are exemplified by the following case reports.

REPORT OF CASES

CASE 1.—A man of forty-four years was referred by one of the major insurance companies for special examination, to determine whether arterial disease was a factor in a long-continued disability. He had no symptoms until August 23, 1933, when, while employed as a tractor operator, the ends of the fingers of the right hand were struck by the steering lever. Many of the details of the history were elicited only after careful questioning at the time of special examination. A few days later, small gangrenous spots appeared on the tips of the index and ring fingers, and finally sloughed in about three weeks. The right middle finger became bluish, swollen and exquisitely tender, so that on September 9, 1933, he was forced to discontinue his employment. On September 20, 1933, "pus" accumulated under the nail and the nail was removed. The entire nail bed then became gangrenous, separating about a month later. X-rays showed a fracture of the terminal phalanx. Healing was so delayed that the terminal phalanx was amputated, and after the stitches were removed the wound edges separated and became infected. On March 8, 1934, the wound was healed, but opened again soon afterward, extruding sequestra and forming small abscesses followed by sinuses. This never again healed, and on August 21, 1934, amputation was done at the proximal interphalangeal joint.

About March 14, 1934, he was attempting to turn a valve when he felt a sharp pain in the index and middle fingers of the left hand. There was no direct injury. In the latter part of April, gangrenous spots appeared on the tips of these fingers, the slough separated, leaving discharging ulcers which did not heal until June 27, 1934. A few weeks later pus accumulated under the nail of the left index finger and the nail was removed. The nail bed became gangrenous, and sloughed in about two weeks, exposing the bone. The bone is still exposed. His left index and middle fingers became cold and cyanotic in cold weather.

On examination it was found that the right radial and brachial pulses were diminished. Both hands blanched on elevation and became ruboric on dependency. The hands were exposed for thirty minutes to a constant room temperature of 16.7 degrees centigrade. The right hand was very cold to the wrist by actual measurement with the dermatherm. There was a diffuse blotchy cyanosis involving the entire hand, worse on the right index finger, which was almost purple. The picture suggested vasospasm. The scars of the previous ulcers were visible. The sutures were still in place from the recent amputation at the proximal interphalangeal joint of the middle finger. The left hand also showed a blotchy cyanosis of the entire hand; especially marked on the index and middle fingers. The hand was cold, but about 2 degrees centigrade warmer than the right. On the tip of the left index finger was an ulcer of its dorsal surface, exposing about one centimeter of the bone of the terminal phalanx. The bone was dark and necrotic. The picture was typical of arterial disease.

The next point in the investigation was to determine whether the evident arterial disease was due to spasm or occlusion. Both hands were exposed to a room temperature of 15.6 degrees centigrade for thirty minutes, and the surface temperatures measured. The left inferior cervical, first and second thoracic sympathetic ganglia were then suffused with novocain by the posterior route. Although complete sympathetic ablation was shown by a typical Horner's syndrome and anhidrosis of the left upper extremity, there was no rise in surface temperature of the digits or change in color. A similar procedure was carried out on the right without effect. This indicated occlusion without spasm. The clinical picture, however, was so suggestive of spasm that it was thought best to check the results. The right ulnar nerve was blocked at the elbow. There was anesthesia in the ulnar distribution, but very little rise in surface temperature of the little finger, although the hypothenar eminence reached almost the normal vasodilatation level. These studies showed that the arterial disease was occlusive in nature and had only a slight element of spasm.

The feet showed absent dorsalis pedis pulsations with blotchy cyanosis similar to that seen in the hands. On the basis of the history and special studies, the diagnosis was made of chronic obliterating arterial disease of all four extremities, worse in the upper extremities; probably thromboangiitis obliterans. The underlying pathology was, of course, not compensable.

COMMENT

It is surprising that with such a typical history and objective findings during an entire year of treatment by good industrial surgeons, arterial disease was not suspected until his last amputation. The realization that severe chronic occlusive arterial disease has been the dominant underlying factor in his disability, and that the outlook is for progression of the disease, is of great significance in the further handling of this case.

* * *

CASE 2.—Another male, age thirty years, was employed in making and bottling preserves. He claimed that his feet and hands were wet almost constantly from the drainage which, he stated, contained acid used in the preservation of the fruit. In about May, 1935, a crack appeared in the tissues of the right heel, which became painful and ulcerated. About four weeks later the right second toe became painful, and within three weeks of the time had become gangrenous. He was treated with suction pressure therapy from July 17, 1935, until August 7, 1935. On September 19, 1935, this toe was amputated. In spite of the continuation of suction pressure therapy, marked swelling appeared on all the toes of the right foot, extending to the ankle, and gangrene appeared in all the toes. There were never any symptoms on the left. He smoked twenty cigarettes daily. On investigating his past history, it was found that in 1929 a small receptacle struck his right great toe, giving him little or no pain. That evening the toe became red and two days later a blister formed, which developed into a discharging ulcer. A few days later the adjacent toe passed through a similar train of events. It took four months for these lesions to heal, and for one year afterward the right great toe became painful in cold weather. This patient presented a claim to the Industrial Accident Commission that his present condition was directly due to his employment and, therefore, compensable.

On examination the upper extremities were normal. The right lower extremity showed a dry gangrenous

process involving all the toes, and extended about one and one-half centimeters on the dorsum of the foot. There was a small ulcer on the inner aspect of the heel about one-half centimeter in diameter. The foot was markedly swollen to the ankle. No foot pulsations were felt. The popliteal pulsation was diminished. There was mild ischemia on elevation and mild rubor on dependency. On the left lower extremity the dorsalis pedis pulsation was diminished, otherwise the vessels were normal. The clinical picture was quite typical of an obliterating arterial disease, involving both lower extremities, especially the right. The further course of the disease confirmed this. About ten days after this examination the lower extremity was amputated at the junction of the upper and middle third of the leg, and pathologic examination showed a severe thromboangiitis obliterans. In the testimony before the Industrial Accident Commission it was shown that the discharges contained no irritating substances which could have been the factor in producing the original ulcer, and that his disability was the end-result of the progress of an underlying chronic arterial disease which had been present for several years, and which had no causal relationship to his employment.

COMMENT

Whenever there is delayed healing of wounds without apparent cause, a history of chronic ulceration or gangrene, or symptoms of arterial insufficiency, and especially when these are associated with objective signs of arterial disease, the patient should be given a complete vascular examination. By this is meant a careful clinical investigation, a carefully controlled test of the part played by the sympathetic nervous system, and tests for the state of collateral circulation of the skin, if indicated. Diminution of arterial pulsations, decrease in local temperatures, ischemia on elevation, rubor on dependency, trophic changes, discoloration, ulceration and gangrene, all give objective evidence of failing circulation. The surface temperature of the part is in direct proportion to the amount of blood flowing through it. By measuring the skin temperature, under controlled conditions, before and after paralysis of the vasoconstrictor fibers, one can determine whether the failure of the circulation is due to arterial occlusion, to pure arteriospasm, or to a combination of both. In the latter instance, one can determine quantitatively the relative proportion of each. Vasoconstrictor paralysis may be induced by various means. We prefer novocain block of the peripheral nerves, using the posterior tibial nerve for the feet, and the ulnar or median nerve for the hands. Whenever there is a question of the reference of pain over sympathtic pathways, the lumbar chain should be blocked directly.

In patients with occlusive arterial disease, the state of the collateral circulation of the skin may be estimated by the local response to intradermal injections of histamin. The lack of appearance of a flare indicates a poor collateral circulation, and warns against the use of such areas as covers for amputation stumps.

If arterial insufficiency is purely or predominately spastic, the treatment is directed to ablation of sympathetic influence. Conservative measures are tried first, and if no relief is afforded, sympathetic ganglionectomy is to be considered. If the disease is predominately occlusive, sympathectomy will give little relief.

Certain selected cases of occlusive disease are benefited markedly by ligation of the main vein to the limb. Our experiences with a small series of cases has been gratifying.

It is felt that suction pressure therapy will have a definite place in the treatment of these cases, but one should be guarded in his prognosis of results until experience with this method becomes more general. In our sympathetic and vascular department, the results of suction pressure therapy have been somewhat disappointing.

IN CONCLUSION

It is hoped that the more general realization of the important part played by diseases of the peripheral arteries in many industrial injuries will lead to a greater coöperation between the industrial and the vascular surgeon, to the benefit of both the patient and the insurance carrier.

450 Sutter Street.

DISCUSSION

VERNON P. THOMPSON, M. D. (2007 Wilshire Boulevard, Los Angeles).—The first portion of Doctor Pearl's paper gives an excellent summary of the surgery of direct injuries to normal peripheral arteries. My discussion will be confined to the second phase of the paper—that of industrial accidents to individuals with preëxisting chronic arterial disease. It would be difficult to compile from any series of cases two more informative than he reports.

The rôle of industrial accidents in precipitating or aggravating symptoms, due primarily to preëxisting occlusive arterial disease, cannot be appreciated except on a basis of thorough clinical study of the circulatory phenomena present. Simple, direct, clinical observations are sufficient in the great majority of cases to establish the adequacy or inadequacy of the circulation.

Can the muscles do a reasonable amount of work without complaints or cramping? If so, the volume flow of blood through them is adequate. Are the feet and lower legs warm, capillary beds pink, the color remaining on elevation, returning quickly after pressure? If so, further encouraging information is obtained. Is the skin elastic, firm, and not atrophic, inelastic, or doughy and edematous? If, on the contrary, impairment of muscular activity, presence of blanching of capillary beds on elevation, poor nutrition of the skin, and muscular atrophy with persistent coolness are present, there is direct evidence of an inadequate blood volume flow to the extremities.

Data as to the various arterial pulsations present or absent, of calcifying changes in the x-rays, are obvious observations to make and record, but do not bear directly on the question of adequacy or inadequacy of the actual capillary blood volume flow. While detailing the more elaborate tests of determining blood flow, whether due to fixed occlusive processes, or due in part to vasospasm, Doctor Pearl has wisely emphasized the easily obtained clinical data which should shorten the waiting period of months, even years, between the onset of symptoms and the diagnosis.

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EMILE HOLMAN, M. D. (Stanford University Hospital, San Francisco).—Experiences such as are recorded in Doctor Pearl's article, illustrate the necessity of careful physical examination of all applicants for positions in industry. Such physical examinations should consider not only the state of the heart and lungs, and the presence or absence of hernia, but most particularly the state of the peripheral vascular system. Absent pulsation in the dorsalis pedis and posterior tibial arteries need not necessarily preclude employment, but a record of an inadequate circulation would be of great help in case of subsequent injury. Moreover, even though symptoms are not present at the time, much can be done to avoid trouble if, in the presence of absent pulsation of peripheral arteries, certain instructions are given to the applicant. He should be warned against injury by ill-fitting shoes, by cutting corns and nails, and by frostbite. If injury does occur, precautions against infection should be carried out immediately: (1) by washing with soap and water; (2) by the application of

a mild antiseptic (not full-strength iodin); and (3) by the application of sterile dressing until healed. A known disability may be easily guarded against; but, if unknown, the loss of a limb may be at stake.

Similarly, in every complete physical examination by a consultant or internist, the state of the peripheral vessels must be recorded. If they are found subnormal or absent, instructions similar to the above may be of untold value to the patient in avoiding trouble. Inadequate circulation may be endurable and without danger if uncomplicated by injury or infection. If infection is added to the disability of inadequate circulation, a vicious circle may be set up which threatens life itself.

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WILLIAM J. KERR, M. D. (University of California Hospital, San Francisco).—There is little to add to Doctor Pearl's discussion. The basic principles of value have been stated, and although many details of the tests may be added, enough have been mentioned to help the clinician in the analysis of the problem in each patient. When patients with circulatory disorders present themselves, it has been most helpful for me to keep in mind the structures and their functions which are important in the maintenance of the normal circulation to these parts. First, comes a careful description and location of the lesions; then a study of all the arteries of the body available for examination as to size, consistency and patency, with pressures and quality and volume of pulsations. The veins and lymphatics should be surveyed also. The nervous system and osseous system may be at fault and should be examined carefully. It may be necessary to determine the element of spasm in the causation of symptoms, and this can be accomplished by the methods mentioned. Usually simple methods are most helpful. Histologic examination of tissues may be necessary and many special studies may be required to determine etiologic factors. When all methods of examination are completed and the cause of the circulatory disturbance has been found, the outlook from treatment is too often very disappointing. Very little result of a permanent nature can be expected by any method of treatment in the progressive degenerative and proliferating diseases of the arteries. The symptoms and signs which are stated to be characteristic of certain vascular diseases of the extremities are usually due to alterations of function, and may be due to many types of primary lesions. The problem in industrial medicine and surgery is a difficult one. We can expect that many patients with faulty arterial supply to the extremities may continue for extended periods without symptoms and, following slight injury, develop serious lesions rapidly. We recognize this principle when we urge great precaution in the care of the feet in the arteriosclerotic and diabetic patient. The problem of industrial hazard is the same as in the cardiac, tubercular, or arthritic patient.

Paralytic and Preparalytic Poliomyelitis.—According to a note in *Public Health Reports,* two states have already instituted a classification for reporting cases of poliomyelitis into paralytic and preparalytic or nonparalytic types. The Department of Public Health of Massachusetts placed this classification in effect at the beginning of the present year. Effective October 20, a similar division was to be reported in the state of Tennessee. The number of cases of preparalytic poliomyelitis which are included in the total reported for that disease will be stated in each weekly report. These are nonfatal cases of poliomyelitis that have not shown definite muscular weakness. Because of the variability and uncertainty in recognition of nonparalytic poliomyelitis, it is believed that for recording and comparing intensity of spread of poliomyelitis only the paralytic cases should be counted when such distinction is possible. Any notable number of nonparalytic cases will be reported separately. There is no intention in this procedure to minimize the importance of the preparalytic or nonparalytic cases from the point of view of the spread of the disease or the necessity for medical care.—*Journal of the American Medical Association,* December 12, 1936.

THE LURE OF MEDICAL HISTORY†

THE HUNTERS IN EMBRYOLOGY*

By A. W. MEYER, M.D.
Stanford University

III **

WILLIAM HUNTER deserves great credit for regarding the decidua as a growth of the lining of the uterus instead of a coagulum. It seems that the idea that it was a coagulum was attributed to William through the text of 1794 to the Gravid Uterus, written by his brother John and Matthew Baillie.[30] The idea hence became current as that of William instead of John, perhaps because the 1794 text appeared later and had a wider circulation than the famous atlas itself. It is noteworthy that even von Baer,[31] in his observations on the development of man, which appeared in 1835, spoke of the Membrana decidua hunteri as a layer of unorganized secretion lying upon and between the elongated villi of the pregnant uterus. Von Baer thought the blood vessels invaded this layer, growing around the villi and forming so complete a plexus that he could not distinguish the arteries from the veins. He believed that this indicated that the relation of the decidua to the uterus is that of an inflammatory exudate to the injured tissues, and added that he hesitated to regard the human decidua as a "proliferation of the mucosa as Seiler recently had done, and shared the more generally accepted opinion that it is a coagulum." In discussing the idea further, von Baer brought together a number of facts which he believed supported a view also held by Purkinje[32] (1834).§

It may have been fortunate that John was probably unaware of the views of "eminent anatomists" referred to by Haller on this matter, unless he could also have known what Aranzi[33] and others had thought, and what Falconnet[34] and

†A Twenty-Five Years Ago column, made up of excerpts from the official journal of the California Medical Association of twenty-five years ago, is printed in each issue of CALIFORNIA AND WESTERN MEDICINE. The column is one of the regular features of the Miscellany department, and its page number will be found on the front cover.

*From the Department of Anatomy, Stanford University.

** Part I of this paper was printed in the November issue, page 420; Part II, in the December issue, page 492.

30 Hunter, William: An anatomical description of the human gravid uterus and its contents, edited by M. Baillie. London, 1794, 4to.; second edition, by E. Rigby, London, 1843, 8vo. (Cited after Bettany.)

31 Von Baer (Karl Ernst): Beobachtungen aus der Entwickelungsgeschichte der Menschen. Aus einem Schreiben an den Herausgeber. Journal für Geburtshülfe, 14:401-417. Leipzig, 1835.

32 Purkinje (J. E.): Das Ei. Anzeige aus dem "Encyclopädischen Wörterbuche der medicinischen Wissenschaften." Herausgegeben von den Professoren der medicinischen Facultät zu Berlin, *Busch, Gräfe, Hufeland, Link* und *Müller.* X. Band. 1834. 8: Journal für Geburtshülfe, 14:375-399. Leipzig, 1835.

§ Purkinje gives the following terms for decidua: Sandifort called it Decidua externa; Haller, Membrana externa ovi; Hunter, M. caduca or decidua; Mayer, Caduca crassa; Osiander, Membrana mucosa; Meckel, maternal egg membrane (mütterliche Eihaut); Chaussier, Epichorium; Danz and others, deciduous membrane (shedding skin); Bojanus, Decidua cellularis and spongiosae; Burdach, Nidamentum; Velpeau. Anhiste; Breschet, Membrana caduque primitive; Seiler, Membrana uteri interna evoluta.

33 Aranzi, Giulio Cesare: De humano foetu, etc. Third edition, Venet., 1587. First edition, 1564 (Rom.) or 1572. (Cited after Fasbender.)

34 Falconnet, Camillus: Non est fetui sanguis maternus alimenta. Paris, 1711. Also in Haller's Disputationes selectae, 1750. (Cited after Needham.)